TWO CASES OF ACUTE ASCENDING PARALYSIS, WITH AUTOPSY.

By John Jenks Thomas, A.M., M.D.,
ASSISTANT TO THE PHYSICIANS FOR DISEASES OF THE NERVOUS SYSTEM, BOSTON CITY HOSPITAL; ASSISTANT NEUROLOGIST TO THE CHILDREN'S HOSPITAL; MEMBER OF THE AMERICAN NEUROLOGICAL ASSOCIATION, ETC.

(*From the Pathological Laboratory of the Boston City Hospital.*)

In 1859 the French physician Landry published his paper describing a disease which, until that time, had been confounded with others. His original description of this disease was that it was an acute paralysis beginning in the legs, ascending to the arms, and later involving the muscles supplied from the medulla. This paralysis was unaccompanied by any marked disturbance of sensation, although he stated that, at times, slight paræsthesiæ might be present. The sphincters were usually unaffected. He also stated that the paralyzed muscles retained their excitability to the faradic current, that the mind remained clear, and that fever was slight or absent. Another criterion which he gave was that after death no lesions were to be found in the nervous system, and that the careful microscopic examination of the cord was negative.

Since that time many cases have been reported as examples of this disease, many of which have varied considerably from the original picture as described by Landry.

The two following cases present a picture very closely resembling the original case described by Landry, and are reported together, because in both cases pathological examinations were obtained and made. I am indebted to Dr. Sears for permission to use the clinical notes of the first case, and to Dr. Folsom for those of the second case; also to Dr. Bullard for permission to use notes which he had made. I also wish to express my thanks to Dr. Councilman, who kindly allowed me to examine the material and aided me in the work.

Case I.—N. G., female, aged thirty-six years; single. She entered the Boston City Hospital on August 29, 1896. She was born in Vermont, and her occupation was that of a housekeeper. One brother died of phthisis. Her mother was said to be very nervous, and died suddenly three years ago of heart disease. The father was alive and well. The patient was never very strong, and lost much time from school. She had diphtheria and scarlatina when a child, and measles and mumps two years ago. Since that time she had been well, until last March, when she began to run down. Two years ago, after the shock of the death of her mother, she had a period of numbness in the right arm, lasting several weeks. This was not accompanied by any loss of strength. Her appetite was good. She denied the use of alcohol and morphine, and drank no tea nor coffee. There was no venereal history. Four years ago she had an ischio-rectal abscess, resulting in a fistula which remained unhealed.

Present Trouble. In March, 1896, she took a severe cold, and since that time had not felt so strong. She had lost twelve pounds, and felt weak, and was easily tired. She had considerable dyspnœa at times, and complained that "the food was heavy on her stomach" and caused distress. The bowels had been much constipated. The appetite was good. The catamenia were regular and without pain. She had never been subject to headache, but about a year ago her eyes pained her, and she procured glasses, and had no trouble after that. She had been much confined to the house. On August 5th she had an attack of indigestion. She went to bed and took salts and calomel. Toward evening of that day she noticed a weakness in her legs, and that she had difficulty in going up stairs. She went to bed feeling perfectly well, except for this weakness, but on waking in the morning she found that she could not move her legs at all. The legs felt numb and dead, and they were somewhat painful to pressure. The bowels had moved once after the calomel, but not after that (paralysis of the rectum ?). There was also retention of urine.

Physical Examination. The patient was bright and intelligent. She was well developed and had good color. The pupils were clear, regular, normal in size, and reacted to light and with accommodation. The external ocular muscles were normal. The tongue was moist, with a white coat, and was protruded in the median line. The pulse was regular, of good strength and volume.

Heart: The area of cardiac dulness was not enlarged, the apex was in the fifth interspace, three and three-quarter inches to the left of the median line. The heart's action was regular. The first sound at the apex was roughened.

Lungs: The resonance and respiration were good throughout. The area of hepatic dulness extended from the sixth rib to the costal border.

The area of splenic dulness was not enlarged.

The abdomen was tympanitic, not tender.

There was slight power of flexion of both legs. The toes and feet could be moved slightly; those of the left foot more than the right. The patellar and plantar reflexes were absent. The sensation was nowhere affected. There was considerable tenderness on pressure in the calves of the legs. In the upper extremities the grasp was weak on both sides, the left more so than the right. What muscular power remained in the arms was greater on the right side. The sensation here was normal, as also on the trunk. There was no tenderness in the upper extremities on pressure.

On August 14th a small area of bronchial breathing was noted just above the angle of the scapula and just outside of the median line in the left back. There was no cough and no expectoration. There had been considerable dyspnœa. The temperature had fallen somewhat, but the respirations had increased in number. The bowels moved only by enemata. The urine was passed spontaneously. Since entrance the strength of the right arm had diminished. On this day she could flex and extend the forearm slightly, but could not raise the arm from the bed. The only movement remaining in the left arm was a slight movement of the fingers. There was absolutely no movement possible of the lower extremities. The tenderness upon pressure in the legs had diminished.

The examination of the urine on August 14th showed it reddish in color; specific gravity of 1.018; alkaline in reaction; chlorides diminished; the urea normal in amount; albumin, a trace. Bile pigments and sugar were absent. The sediment showed quantities of triple phosphates, some pus, a little abnormal blood, a few questionable granular casts, and several large round cells.

On August 18th it was noted that the tenderness in the arms and legs upon pressure had returned. The patient could still flex the left arm feebly. Other movements were impossible. There was incontinence of urine.

A second examination of the urine, made upon August 19th, was as follows: Color, light; specific gravity, 1.017; reaction neutral; chlorides diminished; urea normal; a trace of albumin was present; bile pigments and sugar were absent. The sediment consisted chiefly of pus, with a few small round cells, a few triple phosphates, but no casts were found.

On the 19th there was paralysis of respiration. On the 22d it was noted that she had periodical attacks of difficult breathing, when the throat seemed to choke up and suffocation threatened. She had had two severe attacks of this kind, both of which passed off. All the muscles of respiration were noted upon this day as being inactive except the diaphragm. She complained that she could not get into a comfortable position, and she had to be moved continually.

On the 23d it was noted that there was slight movement possible of the left wrist, and that she could flex and extend the fingers of the left hand. The right arm could be slightly abducted, but could not be moved backward or upward. All movements of the right forearm and wrist were possible. There was complete paralysis of all the muscles of the lower extremities, of the abdominal muscles and the muscles of respiration, with the exception of the diaphragm. There was no paralysis of the face, tongue, or pharynx. The sensation everywhere was normal. There was considerable tenderness in the calves and slight tenderness in the thighs upon pressure. The glands in the neck were enlarged. The liver could be felt at the level of the umbilicus and could be seen to move with the movements of the diaphragm. The patient was unable to cough or to make forced expiration.

On August 24th at 8.30 A.M. the patient was found breathing with difficulty, the face was cyanotic, but she had no difficulty in speaking. The nurse reported that at 7 A.M. the patient had seemed as usual. From 8.30 A.M. the dyspnœa increased gradually, the pulse became weaker, and she died at 11.50 A.M.

CHART.

	Temperature.		Pulse.		Respiration.	
	A.M.	P.M.	A.M.	P.M.	A.M.	P.M.
9th,	.....	102°	....	95	....	24
10th,	101°	101.5	94	95	23	24
11th,	101	101	90	94	24	20
12th,	99.4	99.8	100	100	24	24
13th,	99.8	100.8	100	100	24	28
14th,	100	98.5	100	90	40	28
15th,	98.4	98.4	96	90	32	28

	Temperature.		Pulse.		Respiration.	
	A.M.	P.M.	A.M.	P.M.	A.M.	P.M.
16th,	98.2°	98.4°	80	72	24	24
17th,	98.4	99	72	70	24	24
18th,	99	98	80	84	24	24
19th,	98	99	80	84	28	26
20th,	98.4	100	100	104	25	26
21st,	98.2	98.8	88	100	24	24
22d,	99.2	100	100	110	36	32
23d,	98.6	98.4	114	112	32	28
24th,	98	......	140	......	44	...

Autopsy, August 25, 1896. The body was that of a woman of about forty years of age. Body length 162 cm.; fairly well developed; poorly nourished. Abdomen markedly distended. The feet in the position of extreme dorsal flexion. Fingers sharply and apparently forcibly flexed on the metacarpals. Subcutaneous fat much diminished.

The intestines, particularly the large intestines, were widely distended, containing much gas with considerable feces.

The appendix was 8 cm. long, extending down behind the ileum, the tip overhanging the pelvis; it was free throughout its whole extent.

The heart weighed 220 grammes. The muscle was deep red in color. The valves and cavities were normal.

The left lung was adherent over a small portion of the upper lobe by old adhesions. In the lower lobe of the left lung a large area was apparently completely solidified; on section this was mottled, yellow and deep red; the bronchi in this region were filled with a yellow, purulent material. The right lung was normal except for considerable fluid at the base.

The liver weighed 1020 grammes. It was normal in color, and on section normal in appearance.

The spleen was flabby, of a grayish-red color, on section rather soft, the pulp normal, weight 80 grammes.

The kidneys together weighed 260 grammes. The cortex was much thinned, in parts being from one-third to one-half cm. thick. The whole kidney was deeply injected, the glomeruli standing out as bright red points. The capsule was rather adherent, and some of the cortical substance adhered to it when it was torn off.

On the right side in front of the uterus there was a large, hard, pedunculated tumor. On section this was white and firm. Otherwise the uterus and appendages were normal.

The bladder contained a small amount of thick, yellow, cloudy urine. The mucous membrane of the bladder was much injected. Scattered over the surface were prominent masses of good-sized vessels, and the tissue about these areas was deeply injected.

The aorta had scattered over its surface many small yellow, opaque, prominent areas.

The nerves of the lumbar and sacral plexus, as well as the pneumogastric, on either side, were removed for microscopic examination. No gross change was apparent. The dura of the cord was distended with considerable clear, rather thick, serous fluid. No gross lesions were apparent in the cord.

Bacteriological Report. Cultures from the heart, liver, and spleen were sterile. Those from the kidney gave colon bacillus and liquefying organisms. Cultures from the spinal cord showed liquefying organisms.

The stains used in the examination of these two cases were those of Nissl and Lenhossek for the ganglion cells; Weigert's and Pal's myelin sheath stains, phosphomolybdic acid hæmatoxylin, hæmatoxylin and eosine; Van Gieson's picric acid and acid fuchsin stain; Mallory's methods of staining the neuroglia fibres with gentian-violet, and phosphotunstic acid hæmatoxylin; and Weigert's and Loeffler's stains for micro-organisms; together with Marchi's method of staining for fat. For the peripheral nerves, Marchi's and Weigert's methods and hæmatoxylin and eosin were the stains used.

After hardening, the anterior portions of the gray matter showed darker in color than the remainder of the gray matter of the cord.

Sections of the lumbar cord stained by Pal's method showed the white matter of the cord with low power normal, except that the small bloodvessels entering from the membranes were dilated and filled with blood.

There was no marked degeneration in any of the nerve-tracts. The posterior nerve-roots were normal. The anterior nerve-roots showed very marked degenerations, fully two-thirds of the fibres having disappeared. The posterior horns of the gray matter were perfectly normal, both as to nerve-cells and fibres. The anterior horn on the right side was totally replaced by an apparently homogeneous grayish area. In the left horn the condition was the same, but there remained unaffected a small portion toward the anterior fissure, which was about one-fourth of the total anterior horn.

On examination with a high power there were seen in the white matter of the cord numerous single nerves which were degenerated. Some of them were greatly swollen, the myelin sheath often very thin and more or less broken up, and occasionally there were drops of myelin within the myelin sheath. The posterior nerve-roots showed very few degenerated fibres. Occasionally one was seen where the myelin sheath was thinner than normal. In the anterior nerve-roots a greater part of the nerve-fibres had disappeared. Of those that remained about one-half appeared normal. In the others the myelin sheath was reduced to a mere thread, and large, irregular drops of myelin were present. In the posterior horns of the cord the nerve-fibres and cells appeared unchanged. In the anterior horns the portions which with the low power presented the homogeneous appearance were seen to be made up of a mass of smaller and larger cells, some containing sharply staining nuclei, others with indistinct nuclei, and here and there in the left horn near the borders of the degenerated area were large, irregular masses of myelin and occasional cells staining a homogeneous brown, apparently degenerated nerve-cells, only one or two of which show any signs of a nucleus remaining. Many of these small cells throughout the degenerated area contained irregular drops of myelin staining black, and at the borders of the area were occasional large masses of myelin or remains of a degenerated myelin sheath.

Sections of the lumbar cord stained with hæmatoxylin and eosin showed dilatation and injection of the small bloodvessels extending into the cord, both those from the periphery of the cord as well as those from the anterior fissure. In many of the vessels the perivascular lymph space was entirely filled with large and small lymphoid cells, which also infiltrated the wall of the vessel itself.

The vessels of the posterior column of the cord and of the posterior nerve-roots showed only very slight infiltration of the perivascular space. Many of them were entirely normal. In the anterior nerve-roots about the smaller vessels there was considerable infiltration with round cells, and round cells were seen with greater frequency than normal all through the substance of the nerve-root. These cells were usually small, with small, round, deeply-staining nuclei, but many of them had large, single, faintly-staining nuclei, oval or round in shape, nearly filling the cell body.

The white matter of the cord in the lateral columns showed, aside from the infiltration about the bloodvessels, practically normal appearances. Some of the small vessels contained an accumulation of leucocytes, from which apparently a slight emigration into the surrounding tissue was taking place. This was not generally true. In the majority of the vessels there was no such accumulation of the leucocytes, but there was a small, round-cell infiltration of the vessel walls and of the perivascular lymph spaces. This infiltration about the vessels was more marked in the case of the arteries. Most of these cells were small, with round, very deeply-staining nuclei, almost filling the cell. Others, fewer in number, with slightly granular protoplasm, contained a round or oval granular nucleus which stained intensely. Leucocytes were few in number. There was no hemorrhage about the vessels in the substance of the cord.

The degenerated portions of the anterior horn of the gray matter appeared totally disorganized. Only occasionally in the borders of this area could be seen the remains of nerve-cells. These were small, shrunken, homogeneous-staining masses of protoplasm, occasionally showing indications of processes. In a few of them, one portion, taking the hæmatoxylin more deeply than the rest, showed the indistinct remains of the nucleus.

The degenerated area itself consisted of a disorganized mass of cells and detritus. These cells varied in size, some of them being small, round, lymphoid cells, with round, deeply-staining nuclei, surrounded by a narrow band of clear protoplasm. Other cells were similar to the large lymphoid cell with nucleus rather small in relation to the cell body, and the nucleus divided into a number of granules with more or less reticulum between them. These cells were the plasma cells of Unna. The protoplasm was finely granular, and with methylene-blue stained more deeply than that of other cells. They were found chiefly in the vicinity of the vessels, but also to a certain extent scattered among the other cells. Very rarely there were seen large round cells with irregular, less deeply-staining nuclei similar to those of leucocytes.

The small round cells similar to lymphocytes were comparatively few in number. There were also scattered about a considerable number of large vesicular cells; in some of them the nucleus was round, in others similar to that of a leucocyte. These cells were the ones spoken of below as containing the drops of myelin and fat. There were numerous larger and smaller hyaline masses scattered through this area, sometimes drawn out, sometimes in rounded masses, evidently the remains of nerve-cells and nerve-fibres. One of these masses was found partially surrounded by a large vesicular cell. Occasionally throughout the whole of this area large vesicular cells were seen filled with drops of fat and myelin, as was shown by the sections stained according to Pal and

Marchi. These sections also showed the presence of fat and myelin drops in a large portion of the smaller lymphoid cells. Scattered throughout this area were masses of myelin or fat not contained in cells. Occasionally about one of these drops of myelin could be seen a portion of a degenerated myelin sheath. This same section showed that the degeneration of nerve-fibres in the lateral columns, while involving numerous nerve-fibres, was still moderate in amount. The same was true of the posterior nerve-roots. The anterior nerve-roots showed signs of very extensive destruction of the myelin sheaths.

Sections stained with phosphomolybdic acid showed nearly normal conditions in the white matter of the cord and of the posterior horns of the gray matter. There were almost no nerve-cells in the anterior horn.

Sections stained by Nissl's method showed the presence of perfectly normal nerve-cells in the posterior horns; while in the anterior horn on the right side in one section there were only four nerve-cells, two of which were irregular, round masses of protoplasm containing no granules, with intensely-staining nuclei. The other cells showed the nucleus and nucleolus somewhat more plainly; the granules were fairly distinct, but smaller than normal. But one or two processes could be seen to be given off from the cells, and these could be traced but a very short distance from the cell-body, apparently ending abruptly.

In one of these sections a cell lying in the wall of a vessel showed in its nucleus a karyokinetic figure.

Sections of the lumbar cord stained by Mallory's stain for neuroglia showed no increase in the neuroglia anywhere in the cord, and practically complete absence of the neuroglia fibres in the degenerated areas of the anterior horn.

Sections of the dorsal and cervical cord showed practically the same conditions that were seen in the lumbar regions. The degeneration of the anterior nerve-roots was fully as extensive as in the lumbar sections, while the posterior nerve-roots showed practically normal conditions.

In the dorsal cord the disintegrated area in the anterior horns of the gray matter, the infiltration of the bloodvessels of the cord and of the anterior nerve-roots was fully as extensive as lower down. In the cervical cord, the right anterior root showed only a small area of degeneration in its anterior portion. The left horn showed more extensive degeneration involving the anterior and median portions of the horn from its base down to the centre of the gray commissure. Infiltration about the bloodvessels of this portion of the cord was less extensive than lower down, but many of the vessels were widely dilated and filled with red blood-corpuscles, with comparatively few leucocytes, while the infiltration of the walls of the vessel was extensive in some portions and very little in others.

Sections of the cord at various places stained by Weigert and Loeffler's methods for micro-organisms failed to show the presence of any organisms.

Sections of the sciatic nerve stained by Marchi's method were in general normal. The longitudinal sections showed slight degeneration of the myelin sheath. Cross-sections of the nerve showed similar degeneration, but not very extensive.

Sections of the pneumogastric nerve showed marked fatty degeneration. In the longitudinal sections there was a great deal of fat in the myelin sheath. In addition, the sheath was swollen and in many places had a grayish tinge. The axis-cylinder was also swollen and beaded. In places where the nerve-sheath was most degenerated the axis-cylinder was not apparent. In other places where the myelin sheath was swollen and grayish it was difficult to make out the axis-cylinder. Cross-sections of this nerve showed in places swollen nerve-sheaths without axis-cylinders. Other nerve-fibres showed fat-granules often within the myelin sheath and lying close to the axis-cylinder. The nerves in which the myelin sheath was granular, grayish, and swollen, here showed more evidence of the presence of fat-globules than was shown in the longitudinal sections. In the anterior crural nerve the nerve-sheaths occasionally showed slightly degenerated fibres, but these were less numerous than in the other nerves. The phrenic nerve showed a much less degree of degeneration than the pneumogastric. The nerve-fibres replaced by fat-granules were much less numerous, but in the grayish swollen nerve-sheaths swollen and beaded axis-cylinders were often evident.

Sections of the pneumogastric nerve stained with hæmatoxylin showed the axis-cylinder of many of the fibres distinctly swollen and beaded. The bloodvessels were injected everywhere, and in the sheath of the vessels there were numerous round cells and, in places, slight accumulations of round cells, which often extended from the connective tissue slightly into the nerve.

SUMMARY OF MICROSCOPIC EXAMINATION. 1. Acute inflammatory exudation of the anterior horns of the gray matter, with parenchymatous degeneration of the nerve-cells and processes.

2. Infiltration of perivascular lymph spaces and dilatation of vessels of the anterior horns.

3. Moderate infiltration about the vessels of the posterior horns and of the white matter of the cord.

4. Slight parenchymatous degeneration of the nerve-fibres of the white matter of the cord.

5. Slight degeneration of the posterior nerve-roots and marked degeneration of the anterior nerve-roots.

6. Parenchymatous degeneration and perivascular infiltration of the peripheral nerves.

7. Absence of micro-organisms in sections and cultures.

Anatomical Diagnosis. Broncho-pneumonia of left lung. Chronic, diffuse nephritis. Arterio-sclerosis of aorta. Myoma of uterus. Acute anterior poliomyelitis.

CASE II.—V. F., male, aged thirty-five years, married, was born in Italy, and his occupation was that of a laborer. The patient entered the Boston City Hospital, October 5, 1896. His family history was negative. He had been four years in Boston. He denied the use of alcohol, and, so far as known, had been well and strong up to twelve days before. At that time he began to be troubled by numbness and weakness in his legs. This increased for two days, at which time he was obliged to give up work and go to bed. He had very little pain, and no nausea or vomiting. He was said to have had one attack of dyspnœa on the day of his entrance into the hospital. He ascribed his trouble to exposure to cold and rain. His chief complaint was numbness in the legs.

Physical Examination. The patient was a well-developed and nourished man. The pupils were equal in size and reacted to light. The tongue was protruded straight, and was moist, with a slight coating. The pulse was regular, of good strength and volume. The area of cardiac dulness, the cardiac sounds, and action were all normal.

The resonance over the lungs was good throughout. The respiration everywhere was accompanied by squeaking and sonorous râles, and a few coarse, moist râles.

The areas of liver and splenic dulness were normal. The abdomen was negative. There was marked paralysis of both lower extremities, the only motion possible being a slight flexion of the knees. There was also marked weakness of the muscles of the trunk and neck. The head could be rotated, but not raised. There was a very slight power of contraction of the abdominal muscles, and a marked loss of power in all the muscles of the upper extremities. The arms could be raised very slightly only; the grasp was weak. The patellar reflexes were absent, as were also the reflexes of the arms. The plantar reflexes were present, but very slow. The cremasteric and abdominal reflexes were normal. The sensation for pain was everywhere normal. There was no tenderness along the spine or over the great nerve-trunks.

Examination of the urine made October 6th showed the color normal; the specific gravity 1.025; acid reaction; chlorides increased. Albumin, bile pigments, and sugar all absent. On October 7th it was noted that the patient complained of no pain, and took his nourishment well. There was no retention of urine, but the bowels had not moved. In the night he had an attack of dyspnœa, and had been unable to expel the mucus from his throat. That morning marked conjunctivitis was noted. At 6.30 o'clock that evening he began to have severe dyspnœa, the pulse became weak, and the extremities cold. He was given strychnine subcutaneously, but died at 7 o'clock.

CHART.

	Temperature.		Pulse.		Respiration.	
	A.M.	P.M.	A.M.	P.M.	A.M.	P.M.
Oct. 5th,	98.6	98.6	96	88	24	24
6th,	98	98.4	80	92	20	24
7th,	97.8	97.2	124	120	24	32

Autopsy, October 8, 1896. The body was that of a man about forty years of age. Body small, tolerably well nourished. Rigor mortis extremely well marked.

Scalp normal. Skull of ordinary thickness; dura not adherent; rather hyperæmic. The inner meninges and the brain itself hyperæmic. The gray matter of the brain had a pinkish hue on section. The hyperæmia seemed to be best marked in the great ganglia at the base.

Subcutaneous fat normal in amount. Muscles well developed. The spinal canal was opened in its entire extent, and the cord, together with most of the posterior nerve-roots, removed. A number of the spinal ganglia were removed with cord. The meninges of the cord were hyperæmic. The cord itself was well preserved, somewhat hyperæmic; nothing abnormal in the nerves. Long pieces of each sciatic nerve were removed.

In the posterior portion of both lungs, especially the left lung, and in this also along the anterior lateral border, there are numerous dark areas from which occasionally some pus could be expressed from the bronchi. Others seemed to be due entirely to hemorrhage. In the bronchi there was some muco-purulent exudation.

Heart of ordinary size, weight 240 grammes; valves normal; myocardium very dark.

Spleen, weight 175 grammes; very lax, slightly enlarged; on section, of pale brownish color; Malpighian bodies not visible; trabeculæ slightly increased. The kidneys together weighed 175 grammes. Both of about the same size; both intensely hyperæmic; markings normal; capsule easily stripped off.

Liver, weight 1200 grammes. Dark red; on section, hyperæmic; markings faintly visible. The gall-bladder contained thin, dark bile.

The adrenal glands normal. Pancreas hyperæmic and extremely soft.

The mucous membrane of the entire intestinal canal normal, save just above the ileo-cæcal valve, where there was very slight enlargement of the follicles. The mesenteric glands were enlarged and reddened.

Arteries and veins normal.

Bacteriological Examination. Cultures from heart, liver, spleen, kidneys, meninges of brain, and spinal cord were sterile. Four cultures were taken from the brain, three from the meninges of the brain, and one from the spinal cord. Three of these (two from brain and one from meninges) showed no growth. Dilutions and replants from the others showed nothing but saprophytes (a spore-forming bacillus), a large coccus, and a large diplococcus.

Microscopical Examination. Sections of the lumbar cord stained according to Pal showed no changes in the white sections of the cord. The fibres and nerve-cells of the gray matter appeared normal, the nerve-cells being present in normal numbers and showing no changes by this stain. The nerve-fibres in the gray matter were also unchanged. The bloodvessels in the anterior horn appeared distended and filled with blood. There was no infiltration of their walls, but the perivascular lymph spaces of some of them were dilated. The posterior nerve-roots were normal. The anterior nerve-roots showed degeneration of a considerable number of nerve-fibres.

Sections stained with phosphomolybdic acid showed no diminution of nerve-fibres and no changes in the nerve-cells. The axis-cylinders of the anterior roots were occasionally absent, while others stained poorly.

Sections stained with hæmatoxylin and eosin showed no changes beyond the dilatation of the vessels in or near the gray matter of the cord. These vessels contained generally red blood-corpuscles and only a few leucocytes. There was no infiltration of the walls of the vessels. In others of these vessels there was an accumulation of small round cells, with deeply-staining nuclei.

Sections stained for neuroglia showed no increase of this substance anywhere in the cord.

Sections stained with Nissl's stain showed that in a large majority of the cells of both anterior horns there was a diminution of the protoplasmic granules. In some cells there remained in the periphery of the cell nearly normal granules, while the central portion of the cell showed a slightly yellowish homogeneous appearance dotted with a few very small, irregular granules, with no signs of nucleus or nucleolus. In other cells the nucleus could not be made out, but the nucleolus stained indistinctly, and the whole body of the cell was filled with very fine granules, like dust, with very few or no normal granules. Those which were present were only seen at the periphery of the cell. Other cells showed finely granular detritus at one end of the cell, while the other was clear, showing no granules whatever, with no signs of nucleus or nucleolus anywhere to be seen. The cell-processes even of these degenerated cells appeared normal. The cells seen in other portions of the gray matter showed normal granules and nucleus and nucleolus. In the anterior horn, along with these changed cells, were others which appeared nearly or quite normal, the only thing noticeable in them being an irregularity in the size of the granules.

Sections stained according to Weigert and Loeffler showed no micro-organisms present in the substance of the cord. Sections of the dorsal and cervical cord at various places showed the same changes in the large motor cells of the anterior horns, fully one-half of the cells of the dorsal region of the cord being affected, though to a slighter degree than the cells of the lumbar cord. Changes in these cells again became more marked and involved a greater number of the cells in the cervical portion of the cord.

Sections of the medulla stained by Nissl's method showed no changes in the nerve-cells, the granules being sharp and clear, the nucleoli staining well, and the nerve processes appearing normal. In the sections of the cortex of the brain, in some of the larger nerve-cells the granules were smaller than normal and more or less irregularly scattered throughout the whole cell-body. No cells were seen where the granules had disappeared or where they had been replaced by granular dust. The smaller cells of the cortex appeared normal.

Sections of the dorsal and lumbar spinal ganglia stained by Nissl's method showed the ganglion cells in general quite normal. Occasionally one was seen containing a vacuole, while in all of them the protoplasmic granules were rather small. Otherwise the sections of these ganglia appeared perfectly normal.

Sections of the left anterior crural nerve treated by Marchi's method showed numerous fat-globules within the nerve-sheaths. In places the nerve-sheath was entirely replaced by fat-globules. The axis-cylinder had completely disappeared. Other fibres showed a brownish discoloration of the nerve-sheath, which was more granular and swollen than normal, while the axis-cylinder was swollen and beaded. These changes were seen to a greater or less extent in more than half the fibres in the section.

Cross-sections of the same nerve showed drops of fat in the myelin sheath, in other places occupying the centre of the sheath, the position of the axis-cylinder which had disappeared. Other fibres showed a thin line of fat in the myelin sheath. The nerve-fibres varied somewhat in size, but the greatly swollen ones were comparatively few in number, and in these the axis-cylinder could not be made out at all, the centre staining a homogeneous gray color, or the whole nerve-fibre being represented by a single large globule of fat.

The left sciatic nerve, in longitudinal section, showed the same condition as the anterior crural nerve, but affecting a greater number of fibres, and the fat-drops were much more numerous; seldom was a nerve-fibre seen which was free from them, while the beaded appearance of the axis-cylinder was seen in a greater number of them.

Cross-sections showed comparatively few of the nerve-fibres unaffected by changes. The majority of them were replaced by fat-globules, while most of the remainder were stained a grayish brown.

The right sciatic nerve showed similar changes, which were as well marked and fully as extensive as in the left sciatic nerve, fully three-fourths of the fibres in the cross-sections being more or less extensively degenerated.

Sections of the left sciatic nerve stained with hæmatoxylin showed an increase of the connective-tissue nuclei throughout the nerve, and considerable infiltration of small lymphoid cells in the walls of the vessels and about them, both outside and within the nerve-bundles proper. In cross-sections about one-half of the axis-cylinders could be seen, but many of those present were much smaller than normal, while others were greatly swollen, and in many cases the axis-cylinder could not be made out. Infiltration of the vessel walls was well marked in this section also.

Sections of the left anterior crural and right sciatic nerves stained with hæmatoxylin and eosin showed changes similar to those described in the left sciatic nerve, though in the former nerve the proliferation of nuclei and the infiltration of the vessel walls were much less marked.

In sections of the left anterior crural nerve, stained with Weigert's nerve-sheath stain, the majority of the fibres were seen to retain the myelin sheath, approximately one-third of them only being affected by degenerative processes.

SUMMARY. Microscopic examination showed: 1. Parenchymatous degeneration, varying in extent, of the peripheral nerves, present to a greater or less extent in all the nerves examined.

2. Degenerative changes in the large ganglion cells of the anterior horns of the cord, with destruction and fragmentation of the protoplasmic granules and loss of the nuclei of the cells.

3. The nerve-cells of other portions of the gray matter of the cord, medulla, brain, and spinal ganglia unchanged.

4. No change of the white matter of the cord.

5. Absence of micro-organisms in the tissues.

Anatomical Diagnosis. Broncho-pneumonia and hemorrhage of the lungs. Enlargement of the spleen. Congestion of the kidneys. Congestion of the brain and cord. Parenchymatous degeneration of peripheral nerve and motor cells of cord.

After Landry's original publication, there were published in the same year by Kussmaul some similar observations. Yet neither of these papers attracted much attention until Pelegrino Levi in 1865 published another paper upon this disease, and ascribed the death of the celebrated zoölogist Cuvier, who had died of an acute descending paralysis, to this cause.

In 1871 Bernhardt expressed the opinion that the disease was due to a poison. This conception that Landry's paralysis depended upon an intoxication was accepted by Westphal, who in 1876 characterized the disease as one with a progressive, ascending, finally fatal course, with retention of the electrical irritability of the paralyzed muscles, with negative results upon autopsy. This conception of Westphal was soon very much widened, and numerous cases were reported from all sides, which varied in the widest way from the descriptions hitherto given of the disease.

In 1889 Ross, in a series of articles, maintained that this disease was identical with peripheral neuritis. This view was also held by Nauwerck and Barth, in a paper published by them in the same year, and has been the cause of numerous discussions since. The earlier autopsies gave generally negative results, but, as time went on, those in which pathological changes were found began to be more numerous, and it may be assumed that the previous failures to find pathological changes were in many instances due to imperfect methods of examination.

Again, the great diversity of lesions described can undoubtedly be explained from the fact that many writers included cases which probably belonged in classes by themselves. Many of Ross's cases, for instance, must certainly be considered as cases of peripheral neuritis, and the same is true of many other cases which have been published as Landry's paralysis. On the other hand, cases have been published and included in tables where the lesion was a disseminated or localized myelitis or a general central myelitis. Therefore, in the discussion of the pathological changes in Landry's disease, many cases which have been published under this title have been omitted, for reasons which seem sufficient. No attempt has been made to examine cases where a pathological examination was not made.

Among the cases which have been reported where the pathological examination resulted negatively was the original one of Landry,[1] where there was said to have been congestion of the membranes, but no lesion in the brain or cord. The peripheral nerves were not examined.

In Pelegrino Levi's case[2] the nerve-roots, spinal ganglia, vagi, cord, sympathetic and pneumogastric nerves were normal. In the second case reported by him, that of Baron Cuvier, there was no examination made.

Oulmont and Hayem[3] found in their case the spleen enlarged and a slight injection of the pia in the upper part of the cord; the gray matter of the cord reddened, and congestion of the capillaries. After hardening in chromic acid, there could be found no appreciable alteration anywhere. The muscles microscopically were irregularly striated and in places waxy degenerated.

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[1] Landry. Sur la paralysie ascendante aiguë. Gaz. hebdom., 1859, vol. vi. p. 472.

[2] Pelegriuo Levi. Contribution à l'étude de la paralysie ascendante aiguë, etc. Arch. gén. de méd., 1865, 6 série, vol. i. p. 129.

[3] Oulmont and Hayem. Paralysie ascendante aiguë. Gaz. des hôp., 1867, vol. xl. p. 405.

G. Harley and J. Lockhart Clarke[1] found congestion of the vessels of the cord and of the pia, with a softening of the anterior column, which involved the anterior nerve-roots, and more or less of the gray matter of the cord. The medulla appeared normal.

M. Bernhardt[2] found the brain, medulla, and cord normal. The vagi, sciatic, and sympathetic nerves were also normal. The specimens were examined after hardening in Müller's fluid, and fresh. No cultures were made. There was acute hyperplastic splenitis.

C. Westphal[3] reported four cases. In the first there was bronchitis, and tuberculosis was found in the lungs. The spleen was enlarged. The cord and medulla stained with carmine were normal.

In his second case the spleen was found slightly enlarged; the brain, cord, nerve-roots, ganglia, and muscles were normal; moderate degeneration of one of the crural nerves was found. There were no bacteria present in the blood.

His third and fourth cases probably do not come under the head of this disease of which we are speaking.

O. Kahler and A. Pick[4] found an acute hyperplastic splenitis. The cord was normal. There were adhesions between the spinal dura and pia. The nerves were not examined. The specimens were stained with carmine after hardening in Müller's fluid.

H. Kümmell[5] found enlargement of the spleen. The cord was examined after hardening in Müller's fluid and staining with carmine, hæmatoxylin, and methyl-violet. The cord was normal. The peripheral nerves were not mentioned. There was a bilateral hemorrhage in the medulla, without any infiltration of the tissues in the neighborhood with leucocytes. This patient had died of paralysis in the fourth week of typhoid, and the characteristic lesions of typhoid were found in the intestines.

A. Strümpell,[6] in his case, reports enlargement of the spleen, and that the cord, microscopically, was not pathological. The peripheral nerves were not examined.

In Mann's case[7] the spleen was not enlarged; the cord, medulla, and brain were normal. The peripheral nerves were not examined.

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[1] Harley and Clarke. A Fatal Case of Acute Progressive Paralysis. Lancet, 1868, vol. ii. p. 451.

[2] Bernhardt. Beitrag. zur Lehre von der acuten allgemeinen Paralyse. Berlin. klin. Woch., 1871, vol. viii. p. 561.

[3] Westphal. Ueber einige Fälle von acuter tödtlicher Spinallähmung. Arch. f. Psych., 1876, vol. vi. p. 765.

[4] Kahler and Pick. Zu der Lehre von der acuten aufsteigenden Paralyse. Arch. f. Psych., 1880, vol. x. p. 313.

[5] Kümmell. Zur Lehre von der acuten aufsteigenden Spinalparalyse. Zeitsch. f. klin. Med., 1881, vol. ii. p. 273.

[6] Strümpell. Zur Kenntniss der multiplen degenerativen Neuritis. Arch. f. Psych., 1883, vol. xiv. p. 353.

[7] Mann. Landry's Paralysis. Med. Chron., Manchester, 1887, vol. vi. p. 99.

Greppin,[1] in his case, found the brain, cord, peripheral nerves, the spinal ganglia, and nerve-roots healthy. Cultures were negative.

J. A Ormerod[2] reports three cases. In his first the spleen was large and soft. The brain, sciatic and vagus nerves were normal. The stains used were Weigert's, picrocarmine, and aniline blue-black. The cord, stained with carmine, Pal's stain, aniline blue-black, also showed no changes. In his second case the spleen was also enlarged. The cord, hardened in Müller's fluid and stained in aniline blue-black, showed no changes. The anterior tibial nerve, hardened in Müller's fluid, and the cauda equina, hardened in osmic acid, were both normal. In the third case the spleen is reported as rather larger than normal, weight ten and a half ounces, and soft. The nerves, treated with osmic acid and Müller's fluid and stained with picrocarmine or Pal's stain, showed nothing abnormal. In one vagus nerve, however, some of the fibres seemed to take Weigert's stain more deeply than they ought. The cord, stained with picrocarmine, aniline blue-black, and by Pal's method, was normal.

J. Watson,[3] in his case, reports the membranes of the cord slightly congested, and says that no changes noticeable to the naked eye were present. He also adds that sections of the cord were hardened and examined microscopically, but threw no light upon the pathological changes.

A. Albu[4] reports in his case the spleen of normal size and consistency. The membranes of the cord were injected to a moderate degree. The cord was normal. The brain was rich in blood, and showed small hemorrhages in places. The cord was hardened in Müller's fluid and stained with carmine, nigrosin, and Weigert's stains, and showed nothing microscopically, and the bacteriological and microscopical examination of the crural and axillary nerves was negative. Cultures made from the blood, spleen, and cord were also negative.

Von Leube[5] reported a case in which the cord and peripheral nerves showed no changes. They were hardened in Müller's fluid.

M. Prince[6] reports a case in which the cord was microscopically normal. The nerves of the brachial plexus, stained by Weigert's method, showed no changes.

B. Robinson[7] reports a case in which the spleen was normal. In the brain and spinal cord the bloodvessels appeared somewhat congested, but there was no softening. The cerebral convolutions were flattened, more especially over the right parietal lobe, where there was some bulging. In the right parietal lobe a tumor, two and one-half inches in diameter, firmer than the brain tissue, but rather soft, was found. This was found to be a spindle-cell sarcoma. Sections of the cord stained by Weigert's hæmatoxylin, Delafield's hæmatoxylin, and Van Gieson's stain showed no lesion, either inflammatory, degenerative, or of other nature.

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[1] Greppin. Correspondzbl. für schweizer Aerzte, 1892, vol. xxii. p. 517.

[2] Ormerod. Illustrations of Landry's Paralysis. St. Barth. Hosp. Reports, 1892, vol. xxviii. p. 137.

[3] Watson. Acute Ascending (Landry's) Paralysis. British Med. Journ., 1892, vol. ii. p. 1286.

[4] Albu. Zur Aetiologie der Paralysis ascendens acuta, etc. Zeitschr. f. klin. Med., 1893, vol. xxiii. p. 387.

[5] Von Leube. Diagnose der inneren Krankheiten. Leipzig, 1883, vol. ii p. 143.

[6] Prince. A Case of Landry's Paralysis with Autopsy. Journ. of Nerv. and Ment. Disease, 1895, vol. xxii p. 686.

[7] Robinson. A Case of Landry's Paralysis. Med. Rec., 1895, vol. xlvii. p. 534.

This completes the cases found in the literature where the microscopic examination showed no changes. The cases in which changes have been found, but in the peripheral nerves only, or where the changes in the cord were so slight that no importance was attached to them by the writers, are the following:

Dejerine and Goetz[1] found in the cord, hardened in chromic acid and stained with carmine, no changes except that the vessels were somewhat dilated. In the nerves they found degeneration of the nerve-fibres, with increase of the nuclei of the neuroglia, in some of the anterior nerve-roots.

H. Eichhorst[2] found the spleen small; the brain and cord normal. The optic nerves and the optic chiasm were red in color. The peripheral nerves showed evidences of neuritis. This case, clinically, showed a marked diminution of sensation, with pain and tenderness in the extremities, and probably belongs to the cases of peripheral neuritis, as there were no bulbar symptoms. The case developed three weeks after an attack of malaria.

Dejerine[3] reported a case in which the brain and cord were negative; but there were alterations, which he considered of a parenchymatous nature, in the anterior nerve-roots and in the intramuscular nerves.

Curschmann[4] reports a case in which the spleen was four times the normal size. The intestine showed the characteristic changes of typhoid fever, from which the patient was suffering. The brain was normal in appearance. The pia of the cord was hyperæmic; the cord was said to be otherwise normal, except in the lumbar region, where the gray matter was slightly reddened. Upon microscopical examination of the cord a few small areas were seen in the lateral columns, which showed "a sort of swelling with thinning of the axis-cylinders," but no complete destruction of them. The cultures from the lumbar and dorsal cord were negative. Cultures from the upper dorsal and cervical cord and spleen showed the presence of the typhoid bacillus, which was also demonstrated by inoculations. Bacilli were found in the sections of the cord only in the white substance of the cervical and upper dosal regions.

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[1] Dejerine and Goetz. Note sur un cas de paralysie ascendante aiguë. Arch. de physiol., 1876, 2 série, vol. iii. p. 312.

[2] Eichhorst. Neuritis acuta progressiva. Virch. Arch., 1876, vol. lxix. p. 265.

[3] Dejerine. Recherches sur les lésions du système nerveuse, Paris, 1879.

[4] Curschmann. Bemerkungen über das Verhalten des Centralnervensystems bei acuten Infectionskrankheiten. Verhandlungen des V. Congresses für innere Med., 1886, vol. v. p. 469.

A. Nauwerck and W. Barth[1] reported a very typical case, except that the duration was somewhat prolonged (three months). The cord, hardened in Flemming's solution, as well as the medulla, showed no changes. The peripheral nerves showed a moderate degeneration. The lungs were the seat of a beginning tubercular process.

Ross[2] found the internal, antero-lateral, and postero-lateral groups of nerve-cells normal, while the central and median were diseased. There was "granular degeneration of the nerve network," with disappearance of the ganglion cells and increase of the nuclei, together with dilatation and congestion of the bloodvessels. In his second case he could discover no trace of disease in the cord. In an article published later,[3] in speaking of his first case, he says that after further study he came to the conclusion that the changes in the gray matter of the spinal cord were a minor matter.

C. Eisenlohr,[4] in his case, reports the spleen very large, containing no infarctions. In the ileum there were hemorrhages and swelling of the follicles, with enlargement of the mesenteric glands. The brain, cord, and medulla, stained with borax-carmine, nigrosin, and Weigert's stain, showed no changes. There was degeneration of the bulbar and peripheral nerves. Cultures were negative as regards bacteria, as well as smears upon cover-glasses, and the sections.

In his second case he found abundant serum in the spinal canal and the pia slightly œdematous. The spleen was not enlarged, but there was slight swelling of the mesenteric glands and of the follicles of the ileum, where were also found tubercular ulcers. The cord in the eleventh and twelfth dorsal segments showed swollen axis-cylinders in the lateral columns and degenerative products of the medullary substance. The gray matter was normal. He described this as an acute myelitis. The stains used were nigrosin, carmine, picrocarmine and hæmatoxylin. The right vagus was slightly degenerated; the left one normal. The lower cervical and upper dorsal anterior roots showed signs of degeneration. The anterior roots of the lumbar enlargement showed an increase of cells of the connective tissue and adventitia, but no degeneration. There was no degeneration present of the sacral roots. The right tibial nerve was degenerated. Cultures showed two organisms. The first was found in the cord, the spleen, the sciatic nerve, and was the staphylococcus cereus albus. The second organism was found in the spleen and sciatic nerve only, and was the staphylococcus pyogenes aureus. No tubercle bacilli were found. In the sections there were found a long and short bacillus and a coccus.

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[1] Nauwerck and Barth. Zur path. Anatomie der Landry'schen Lähmung. Zieg. Beiträge zur path. Anat. und allg. Path., Jena, 1889, vol. v. p. 1.

[2] Ross. Diseases of the Nervous System, second edition, New York, 1883, vol. i. p. 908.

[3] Ross. Peripheral Neuritis, Med. Chron., Manchester, 1889, vol. x. p. 376.

[4] Eisenlohr. Ueber Landry'sche Paralyse. Deut. med. Woch., 1890, vol. xvi. p. 841.

Hun[1] reports in his case a slight cerebral and spinal meningitis, with infiltration of the walls of the spinal veins. Aside from a degeneration of some fibres of the anterior roots of the cauda equina, the nervous system was normal. No bacteria were found in the tissues, and cultures made from them were negative.

E. Centanni[2] reports in his case that the vessels in the nerves were engorged and the walls were somewhat infiltrated. The nerve-fibres were not much altered. He designates the process as a slight degenerative interstitial neuritis. The cord showed congestion in the vessels of the pia, exudation about the central canal, and some atrophy in the peripheral zone of the cord. Other parts were normal. In the peripheral nerves he found a slender bacillus, with rounded ends, without spores or characteristic grouping. These were usually present in the endoneural lymph spaces. These bacilli were not found elsewhere. No cultures were made.

Giuzetti[3] found the spleen large and soft. There was some disintegration of the nerve-fibres of the cord, which was most marked in the lumbar region; absent in the medulla. The ganglion cells were cloudy, with indistinct nuclei. About the vessels there was a ring of granular substance not staining like fibrin; there were also recent punctiform hemorrhages in the medulla and pons. There was degeneration of both anterior and posterior nerve-roots, but most marked in the anterior ones. In the peripheral nerves there were multiplication of the nuclei and infiltration of some of the vessels. No bacteria were seen in the sections. Cultures made from the brain, cord, sciatic nerve, blood, spleen, mesenteric glands, and urine were all negative, except two tubes from the sciatic nerve and one from the cord, upon which grew a chromogenic bacillus. A rabbit injected with an emulsion of the spinal cord and spleen gave negative results.

The following are the cases found in the literature in which the changes in the cord were marked, whether or not degeneration of the peripheral nerves was found:

P. Baumgarten[4] reported in 1876 a case which is interesting as having been the first in which bacteria were found. These bacilli he identified as the anthrax bacillus. They were found in the lungs, in the spleen, the spinal cord, and the blood. Cramer, in his review of the literature of Landry's paralysis, questions whether this identification of the bacillus found would be accepted at present. Baumgarten found a hyaline substance in the gray matter of the cord, in the commissure, the anterior fissure, and in the perivascular spaces, which he considered an inflammatory exudation. The vessels were distended with blood, and some were filled with pus-corpuscles.

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[1] Hun. The Pathol. of Acute Ascending (Landry's) Paralysis. New York Med. Journ., 1891, vol. liii. p 609.

[2] Centanni. Ein Fall von Landry'scher Paralyse. Ziegler's Beiträge zur path. Anat. u. allgem. Path., Jena, 1890, vol. viii. p. 358.

[3] Giuzetti. Riform. Med., 1894, vol. ii. pp. 5, 19, and 31. Quoted by Bailey and Ewing.

[4] Baumgarten. Ein eigenthümlicher Fall von Paralysie ascendante aiguë mit Pilzbildung im Blut. Arch. f. Heilkunde, 1876, vol. xvii. p. 245.

C. Eisenlohr[1] found in his case the spleen enlarged and soft. The gray substance of the spinal cord was hyperæmic. After hardening in Müller's fluid the cervical cord showed exudation about the central canal and the vessels of the gray matter and in the anterior fissure. The nerve-cells were filled with granular pigment. These changes diminish both above and below, but the same appearances were found in the upper lumbar cord. The medulla showed cellular infiltration about the vessels, and the ganglion cells were swollen and shining, and here and there were capillary hemorrhages. The peripheral nerves were normal.

Schulz and Schultze[2] reported a case which is probably to be regarded as transitional between the typical cases of Landry's paralysis and the paralysis of Duchenne, in that the course was rather long (sixty days). They found degenerative processes in the lateral and pyramidal tracts, as also in the posterior columns. There was infiltration about some of the vessels. There were alterations in the gray matter in the anterior horns, particularly in the lumbar region; the ganglion cells were swollen, granular, and vacuolated. Swollen axis-cylinders were seen in the gray matter and also in the anterior roots. The same changes, but less intense, were found in the dorsal and cervical cord. In the posterior nerve-roots of the lumbar and cervical cord there was seen an occasional swollen axis-cylinder. These abnormal axis-cylinders were also seen in the facial nucleus; otherwise the medulla was normal. There were no bacilli found in the blood. They summed up the changes as consisting of a myelitis of the motor tracts and of the anterior gray substance of the whole cord, and of the lower part of the medulla. There were slight degenerative changes found in the sciatic, ulnar, and peroneal nerves.

Among these cases, in which changes more or less extensive were found in the spinal cord, we must mention two cases reported by Aufrecht.[3] There the changes found seemed more like a localized myelitis than the changes in the cases already quoted. In the first case, in the medulla, below the olive, the central part for the distance of a quarter of an inch was cloudy. Microscopically, numerous large fat-granule cells were found. In the second case, at the upper and middle third of the cervical cord over a space of about 2 mm. in length, were found several small spots, the size of the head of a pin, which upon microscopic examination was seen to be filled with granular detritus, a few red blood-corpuscles, and bodies resembling corpora amylacea.

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[1] Eisenlohr. Ein Fall von Paralysis ascendens acuta. Virch. Arch., 1878, vol. lxxiii. p. 73.

[2] Schulz and Schultze. Zur Lehre von der acuten aufsteigenden Paralyse. Arch. f. Psych., 1882, vol. xii. p. 457.

[3] Aufrecht. Pathol. Mittheilungen. Magdeburg, 1881.

Roussel[1] found vascular lesions in the cord, with degeneration of the ganglion cells. The peripheral nerves were not examined.

J. Hoffmann[2] found the membranes of the brain and cord much congested. In the medulla there was infiltration of the vessels of the meninges and nerve substance. In the corpus restiformis and pyramids there were occasionally swollen axis-cylinders. There was slight extravasation of the red corpuscles throughout the whole of the medulla. The infiltration in the cervical and dorsal cord was more marked. In the lateral columns of the cord there were small groups of swollen axis-cylinders. The same thing was true of the anterior roots. The ganglion cells were shiny and swollen, with indistinct nuclei. Small hemorrhages were seen in the cord, and particularly in the gray substance in the cervical and dorsal cord. The crural and left facial nerves were normal. The right facial nerve showed degenerated fibres. The examination of the blood for bacteria was negative.

Immermann[3] reports a case in which the patient died of pneumonia four weeks after the development of the paralysis. The brain, peripheral nerves, and muscles showed nothing. The anterior horns of the lumbar, dorsal, and cervical cord showed numerous reddish spots. On examination, the vessels were found filled with red corpuscles, and granular cells infiltrated the perivascular spaces. The ganglion cells were replaced in part by hyaline masses. The white substance of the cord was not affected.

Ketli[4] reported a case in which he saw changes in the spinal cord which were those of acute anterior poliomyelitis.

Iwanow[5] found the vessels of the spinal cord enlarged, as well as the capillaries of the gray matter, especially in the anterior horns. There were some capillary extravasations. The walls of the vessels were infiltrated. Similar changes were seen about the central canal, where there was also a fibrinous exudation. The nerve-cells appeared cloudy and swollen, and many of the nuclei were lost. The cord was hardened in Müller's fluid. The examination of the cord for bacteria by Gram's stain resulted negatively.

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[1] Roussel. Arch. de med. nav., 1883, vol. xxxix. p. 370. Quoted by Bailey and Ewing.

[2] Hoffmann. Ein Fall von acuter aufsteigender Paralyse. Arch. f. Psych., 1884, vol. xv. p. 140.

[3] Immermann. Ueber Poliomyelitis ant. acuta und Landry'sche Paralyse. Neurolog. Centralbl., 1885, vol. iv. p. 304.

[4] Ketli. Wien. med. Blatt., 1887, vol. x. p. 250. Quoted by Bailey and Ewing.

[5] Iwanow. Zwei Fälle von acuter aufsteigender Spinalparalyse. St. Petersburg. med. Woch., 1888, vol. v. p. 393.

J. J. Putman[1] also found acute hyperplastic splenitis. The peripheral nerves showed signs of neuritis, and there was infiltration of the nerve-roots. The bloodvessels were crowded with blood. The perivascular spaces and the central canal of the cord contained a "moderate number of cells." The nerve-cells were essentially normal. In the white matter of the cord an occasional enlarged axis-cylinder was seen. In the medulla the vessels were engorged, and there was infiltration of the perivascular spaces, and occasionally slight hemorrhages.

R. T. Williamson[2] reports a case in which the paralysis began in the right hand, extending later to the left, and then to both legs. There was no disturbance of sensation; reflexes were absent; there was rapid atrophy of the muscles. After the first ten days the patient began to improve slightly, but died suddenly from some unknown cause. In the examination of the cord, in the outer part of the anterior horns, the gray matter was found infiltrated with small round cells, and some larger cells evidently containing myelin. The bloodvessels in the neighborhood were dilated and the perivascular spaces filled with round cells. There were no real hemorrhages. The nerve-cells were shrunken and had lost their processes. There was an absence of the normal nerves seen in the anterior horns, though these were present in the other parts of the gray matter. The remainder of the cord was normal, except that the bloodvessels were dilated, and there were infiltrations about some of them. There was an atrophy of the anterior nerve-roots. The posterior nerve-roots were normal. The pia was normal except for dilatation of its vessels and occasional round-cell infiltration at the anterior part of the cord.

E. Klebs[3] reported a case in which there was tubercular pericarditis from a small cheesy bronchial gland. The only change found in the nervous system was a hyaline thrombosis of the central arteries of the cord, with microscopical hemorrhages about these vessels and about most of the nerve-cells, consisting of a moderate or a greater number of round cells, with single large nuclei. Klebs ascribed this thrombosis to an acute infection.

Hlava[4] found the brain and cord normal macroscopically. Upon microscopical examination the gray substance of the cord from the lumbar region up to the medulla, the pons, and corpora quadrigemina was the seat of small cell infiltration and hemorrhages in places. The foci were chiefly in the anterior and posterior horns and also in Clarke's column. The white substance was intact. The vessels everywhere were dilated. In the medulla the infiltration involved chiefly the nucleus of the vagus, hypoglossus and glosso-pharyngeus, and also the olives. The sciatic and the ulnar nerves were examined. In both there were numbers of so-called mast-cells in the medullary sheath, but nowhere was there degeneration.

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[1] Putman. A Case of Acute Fatal Neuritis of Infectious Origin. Boston Med. and Surg. Journ., 1889, cxx. pp. 159 and 187.

[2] Williamson. The Early Changes in the Spinal Cord in Acute Ant. Poliomyelitis of the Adult. Med. Chron., Manchester, 1890, vol. xii. p. 454.

[3] Klebs. Ueber Landry'sche Paralyse. Deutsch. med. Woch., 1891, vol. xvii. p. 81.

[4] Hlava. Examen histologique d'un cas ayant trait à une femme décédée sous les symptomes de la paralysie de Landry. Arch. boh. de méd., 1891, vol. iv. p. 270. Ref. in Schmidt's Jahrbuch, 1891. vol. ccxxxii. p. 244.

Zusch[1] reports a case in which a change in the color of the white substance of the cord was evident—a change which "would indicate an acute myelitic process." The cord seems to have been examined macroscopically only. The paralysis seemed to be the sudden outcome of a very marked stage of chronic alcoholic poisoning.

E. Leyden[2] reports a case where the spleen was small and firm. There was broncho-pneumonia. The blood contained no bacteria. There was neuritic atrophy in the peroneal nerves, in the right radial, and in the recurrent nerves. The cord, hardened in Müller's fluid, showed some thickened and swollen nerve-fibres, with poorly-staining sheaths. These were most abundant in the lateral columns of the dorsal cord, diminishing above. Between these large fibres were small ones, which Leyden thought compressed. Deiters' cells were enlarged, swollen, with clear nuclei, but not increased in number. There were no compound granule cells. The ganglion cells of the anterior horn were swollen and cloudy, so that it was difficult to make out the nucleus, and the nerve processes were swollen. In a considerable number of these cells vacuoles were seen.

W. Oettinger and G. Marinesco[3] reported a case in which the dorsal and lumbar cord was very soft, with injection of the pia; above, the cord was firmer. The medulla and brain were congested and contained small hemorrhages. The changes in the cord followed the vessels of the anterior fissure principally. The vessel-walls were infiltrated, and the vessels themselves contained thrombi. Occasionally there would be a rupture of the vessel with a small hemorrhage in the gray matter. The nerve-cells showed loss of the protoplasmic granules, rupture of the cell processes, and also true atrophy. The changes were less marked in the cervical cord, where they were practically limited to the anterior horns. The same was true of the medulla, where, however, the affection of the nerve-cells was less. Bacteria, mostly streptococci, were seen in the leucocytes and occasionally in some of the nerve-cells, and a diplococcus was also seen. No cultures were made.

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[1] Zusch. Inaug. Diss. Jena, 1891. Quoted in Sajous' Annual of Universal Med. Sci., 1896, vol. ii. B. 18.

[2] Leyden. Ueber mult. Neuritis und acute aufsteigende Paralyse nach Influenza. Zeitschr. f. klin. Med., 1894, vol. xxiv. p. 1.

[3] Oettinger and Marinesco. De l'origine infectieuse de la paralysie ascendante aiguë, etc. Sem. méd., 1895, vol. xv. p. 45.

G. Ballet and A. Dutil,[1] in their case, reported that microscopic examination of the cord stained with hæmatoxylin and eosin showed an enormous dilatation of the vessels of the cord, especially in the anterior horns. The perivascular lymph spaces were infiltrated with leucocytes, and there were occasional hemorrhages. Nissl's stain showed pronounced degeneration of the cells of the posterior horns, of the columns of Clarke, and particularly of those of the anterior horns. These changes consisted of the disappearance of the protoplasmic granules, a swelling of the cells, the separation or rupture of the processes, disappearance of the nucleus, and granular atrophy. These changes were less marked in the medulla. The peripheral nerves showed beginning degeneration. There was little change in the muscles. No bacteria were obtained from cultures, nor were they present in the sections. There was extensive fatty degeneration of the cells of the liver.

P. Marie and G. Marinesco[2] reported a case in which the veins of the pia of the cord were injected. On section the anterior horns were soft and injected. The medulla was more normal, but here there were very evident hemorrhages in the gray substance of the floor of the fourth ventricle and among the root-fibres of the pneumogastric, and occasionally among the hypoglossal fibres. In the brain there were slight pin-point hemorrhages in the gray matter. On microscopic examination of the cord the anterior horns were found to be made up of mononuclear leucocytes, so that the appearance was almost that of an abscess. The leucocytes were disposed chiefly about the bloodvessels. The posterior horn showed only slight infiltration and congestion of the vessels. The nerve-cells, where not completely destroyed, were swollen, and showed loss or fragmentation of the protoplasmic granules, and rupture of the processes from the cells. In the cervical region these changes were less marked, and the same was true of the medulla. A bacillus from 5μ to 12μ in length, articulated end to end, with a clear space between them, with slight enlargement of the free ends, was found in the vessels of the anterior horn, and rarely in the nerve-cells. In the lumbar region this bacillus was not found, but a much shorter organism presenting the form of a "diplo-bacterium." No cultures. No inoculations. The peripheral nerves seemed normal.

Remlinger[3] reports a case in which he found dilatation and inflammation of the vessels of the anterior fissure, and particularly of the branches to the anterior horns. The leucocytes were more numerous in the vessels than normal. He speaks also of the rupture of the cell-processes of the ganglion cells. Nissl's method was used. In the anterior horn in spaces between the large cells, probably in the lymph spaces, he found chains of streptococci. There were no bacteria within the nerve-cells. There were no hæmatozoa in the vessels or out of them. The medulla and peripheral nerves were not examined. Pure cultures of streptococcus were obtained from the cervical, dorsal, and lumbar cord. The blood from the peripheral vessels was sterile.

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[1] Ballet and Dutil. Paralysie ascendante aiguë symptomatique d'une myélite diffuse ascendante. Bull. et mém. de la soc. méd. des hôp. de Paris, 1895, 3e série, vol. xii. p. 684.

[2] Marie and Marinesco. Sur un cas de paralysie de Landry, etc. Bull. et mém. de la Soc. méd. des hôp. de Paris, 1895, 3e série, vol. xii. p. 659.

[3] Remlinger. Sur un cas de maladie de Landry, etc. Compt. rend. de la soc. de biol. Paris, 1896, 10e série, vol. iii. p. 376; also Méd. mod., Paris, 1896, vol. vii. p. 209.

P. Bailey and J. Ewing[1] in their case reported the spleen as enlarged. They found congestion of the bloodvessels of the cord, with exudative inflammation in the anterior horns of the cord and in the medulla. There were vascular and exudative changes in the motor cortex, the basal ganglia, and the cerebellum. There were degeneration of the nerve-cells and vascular changes in the nerve-roots. The peripheral nerves were not examined.

J. Eichberg[2] in his case found congestion of the anterior horns of the cord. There were hemorrhages into the anterior horns, and the tissues about the vessels were infiltrated with round cells. In the lumbar region the process amounted practically to an abscess. In this portion there were no ganglion cells. Higher up in the cord some of the ganglion cells remained, but were altered and surrounded by inflammatory exudation. The alterations were shown by the difference in staining with aniline blue-black, Weigert's, and other stains. There were no changes in the white matter except congestion of the vessels. The membranes of the cord were congested. The anterior nerve-roots were normal. The brain and the peripheral nerves were not examined.

Diller and Meyer[3] reported a case of Landry's paralysis which was normal except that the course was somewhat prolonged (fourteen weeks). They found a diffuse atrophy in the pyramidal tracts, with hyperæmia of the crossed pyramidal tract and increase of the neuroglia. The axis-cylinders of the pyramidal fibres were slightly rarefied. In the anterior horns there was marked pigmentation of the ganglion cells. The nerve-roots were normal. The medulla, brain, and peripheral nerves were not examined.

R. Van der Velden[4] reported a case which is not perfectly typical. In it he says that the legs were stiff, and there was a loss of the electrical excitability of the muscles. The cord showed swelling of the axis-cylinders, with loss of the myelin sheath in various small areas, chiefly in the anterior and lateral columns. There was also some exudation about the vessels.[1]

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[1] Bailey and Ewing. Contribution to the Study of Acute Ascending (Landry's) Paralysis. New York Med. Journ., 1896, vol. lxiv. pp. 1 and 41.

[2] Eichberg. A Case of Ascending Paralysis, etc. Med. Rec., New York, 1891, vol. xxxix. p. 226.

[3] Diller and Meyer. A Case of Landry's Paralysis with Autopsy. THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1896, p. cxi. p. 404.

[4] Van der Velden. Ein Fall von acuter aufsteigender spinal Paralyse. Deutsch. Arch. f. klin. Med., 1877, vol. xix. p. 333.

From a cursory review of the changes found in these autopsies one would almost think that any possible change of the nervous system could cause Landry's paralysis, or no changes at all be required to produce the symptoms. It would hardly seem that it were merely accidental that so many of the cases with negative pathological examination were among the first reported. Without doubt many of these cases if examined with the present methods would have shown definite pathological processes, so that it could scarcely be maintained by any one at the present day that Westphal's criterion of a negative pathological result is necessary in this disease.

Leyden[2] divides Landry's paralysis into: 1. A bulbar or medullary form. 2. A neuritic form. The first, he says, is caused by a process in the cord which extends to the medulla, and is essentially a myelitic process. The other form he thinks is a purely neuritic one, and in this case he states that changes in the electrical reactions of the nerves and muscles often occur, so he says to the process in the second form of the disease, a polyneuritis, there is joined a parenchymatous œdematous process in the spinal cord, which extends to the medulla and, arriving in the neighborhood of the vital nerve-centres, causes death. He also calls attention to the fact that multiple neuritis in its ordinary course shows several essential differences from Landry's paralysis, and mentions the fact that it usually requires less time in which to develop, that there is but a slight tendency to spreading, and no such constant inclination to affect the medulla. He might also have called attention to the fact that multiple neuritis, when extending, is much more apt not to ascend in so regular and orderly a manner as Landry's paralysis.

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[1] The case of Varnali, in the Romania med. Bucuresci, 1896, vol. iv. p. 20, has been omitted because we were unable to obtain access to the journal; the case of Bristow and Horsley, Brit. Med. Journ., 1888, vol. ii. p. 1110, quoted by Ross, because it was evidently rabies.

The following cases, with autopsy, have also been omitted for various reasons—a few because the pathological examination was indefinite; most of them because the cases were not typical of Landry's paralysis.

Leyden. Allgem. Zeitsch. f. Psych., 1875, vol. xxxii. p. 537. Leroy d'Etiolles, 2d part, p. 97. Quoted by Ross.

Ollivier. Traité des maladies de la moelle épinière, 1837, 3d ed., p. 51. Quoted by Ross.

Kussmaul. Zwei Falle von Paraplegie, etc. Erlangen, 1859.

Leudet. Paralysie ascendante aiguë. Gaz. des hôp., 1861, vol. xxxiv. p. 229.

Bablon. Paralysie ascendante aiguë. Gaz. hebdom., 1861, 2e série, vol. i. p. 806.

Leudet. Sur les troubles des nerfs périphériques. Arch. gén. de méd., 1865, 6e série, vol. i. p. 525.

Bourdillat. Paralysie gén. consécutives a la rougeole. Gaz. des hôp., 1868, vol. xli. p. 5.

Chalvet. Thèse de Paris, 1872; also Gaz. des hôp., 1871, vol. xliv. p. 369.

Duchenne. De l'électrisation localisée, 1872, 3d ed., p. 445. Quoted by Ross.

Gombault. Un cas de paralysie spinal. Arch. de phys., 1873, vol. v. p. 80.

Cornil and Lepine. Soc. de biologie, 1873, vol. v. p. 206.

Calestri. Gaz. Lomb., 1874, vol. xxxiv. p. 20; also in Schmidt's Jahrbuch, 1875, vol. clxviii. p. 18.

Leyden. Ueber Poliomyelitis und Neuritis. Zeitschr. f. klin. Med., 1880, vol. i. p. 414.

Fox. A Case of Acute Ascending Paralysis. Brain, 1880, vol. ii. p. 418.

Finny. A Case of Acute Ascending Paralysis. British Med. Journ., 1882, vol. i. p. 732.

Roth. Neuritis disseminata acutissima. Correspondenzblatt für schweizer Aerzte, 1883, vol. xiii. p. 317.

Vierordt. Multiplen degenerativen Neuritis. Arch. f. Psych., 1883, vol. xiv. p. 678.

Wood and Dercum. Acute Ascending or Landry's Paralysis. Ther. Gaz., Detroit, 1885, vol. ix. p. 157.

Buck. A Case of Landry's Paralysis. Lancet, 1885, vol. ii. p. 12.

Sondeykine. Arch. de Neurol., 1886; also in Centralbl. f. klin. Med., 1887, vol. viii. p. 181.

Pitres and Vaillard. Paralysie ascendante aiguë. Arch. de phys., 1887, 3e série, vol. ix. p. 149.

Lunz and Mamurowski. Quoted in Neurol. Centralbl., 1890, vol. ix. p. 696.

Pribytkow. Soc. of Neuropathologists and Alienists at the Moscow University, Sept. 24, 1893. Neurolog. Centralbl., 1894, vol. xiii. p. 716.

Vranjican. Fall von acuter multipler Neuritis. Wien. klin. Woch., 1895, vol. viii. pp. 485 and 511.

Stephens. Landry's Paralysis, Glasgow Med. Journ., 1896, vol. xlv. p. 5

[2] Leyden. Zeitsch. f. klin. Med., 1894, vol. xxiv. p. 26; and Nothnagel's Path. und Therap., 1897, vol. x. p. 445.

Jolly[1] holds practically the same view, but thinks, in addition, that under certain circumstances the cause which produces the injury to the nerve function can act without leaving behind it demonstrable microscopical alterations.

In this connection it is well to remember that Dejerine and Sotta,[2] in a case of chronic polyneuritis of fourteen years' standing, demonstrated pathological changes in the cells of the anterior horn. Ballet and Dutil[3] also reported a case of polyneuritis in which they found the protoplasm of the cells of the anterior horn stained by Nissl's method, homogeneous and swollen, without nuclei or processes. In others the granules were diminished in number, the nuclei were irregular in shape and often at one side of the cell, and there was loss of the nucleolus.

Landry's paralysis must, from its clinical symptoms, necessarily be an affection primarily of the motor neuron of the first order, without involvement of the sensory nervous apparatus. Inasmuch as this motor neuron is both within and without the spinal cord, it is not so strange as it seems at first sight that changes have been missed in the one place or in the other. That changes do occur in the motor cells of the anterior horn in cases of peripheral neuritis can no longer be doubted. The question which concerns us is whether a disease which is purely motor in type should be classed with the ordinary cases of peripheral neuritis in which sensation is usually affected. Certainly Leyden was justified, by the exclusive motor paralysis, the rapid course, and frequent fatal termination of this disease, in separating it from the cases of polyneuritis of more chronic course, with marked pain, sensory disturbances, tenderness of nerve-trunks, and favorable prognosis.

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[1] Jolly. Berlin. klin. Wochenschr., 1891, vol. xxxi. p. 281.

[2] Compt. rend de la Soc. de biol., 1896, 10e série, vol. iii. p. 193.

[3] Ballet et Dutil. Sur un cas de polynévrite avec lésions médullaires. Bull. de la Soc. méd. des hôp., 1895, 3e série, vol. xii. p. 818.

That Bernhardt and Westphal were correct in their supposition that the disease is produced by an intoxication seems to be the general opinion. That the pathological changes found should vary in intensity and extent with the severity of this intoxication need not surprise us. The first of the two cases reported in this paper shows that acute ascending paralysis, the affection being practically altogether of the motor neurons, may find its cause in an inflammatory process, most marked in the anterior horns of the spinal cord, with marked changes in the axis-cylinder throughout the remainder of the course of this motor neuron. The case of Schulz and Schultze seems to occupy a middle ground between these two forms of paralysis. From Duchenne's paralysis these cases are characterized by their more rapid course, and probably also by a slighter tendency of the process to involve the posterior roots and the sensory functions of the cord; from disseminated myelitis by the small section of the cord attacked in its transverse section, and by the anterior horns being affected throughout the whole length of the cord. Clinically, the cases should differ also, as certainly the cases of Leyden and Van der Velden, which are probably of this nature, differ from the typical cases of Landry's paralysis.

Practically these cases come very near the cases of acute anterior poliomyelitis of children, differing from them in that the process is much more generalized over the motor tracks both of the cord and medulla, this latter, as is well known, being seldom affected in the infantile form. So close, indeed, is the resemblance, that Bailey and Ewing, in their paper, have included in their tables six cases reported as acute anterior poliomyelitis occurring in children, because of the extent of the paralysis and the presence of bulbar symptoms which were severe enough to cause death. Practically, one may admit that they were justified in classing these cases with the severe type of Landry's paralysis, such as theirs, that of Marie and Marinesco, that of Oettinger and Marinesco, and the first case reported in this paper. Probably the case of Eichberg also belongs here. In this severer form the process consists of a dilatation of the vessels, with round-cell infiltration of their walls, accompanied by degenerative changes of the motor cells of the anterior horn and their processes. If the pathological process is still more acute the anterior horn may almost resemble an abscess; the normal structure of the gray matter being replaced by an inflammatory product, consisting of large and small lymphoid cells, of compound granule cells (vesicular leucocytes), and the detritus of the degenerated nerve-cells and processes. At times bacteria of different varieties may be present in this inflammatory process itself. More frequently, as in the cases reported in this paper, the examination of the sections for micro-organisms and of the cultures is negative.

The second case seems at first glance to differ entirely from the class of cases which we have just been considering. The examination of the cord by the ordinary methods shows practically no changes, and we should probably not go far wrong if we assume that this represents one of those cases which, in the earlier reports, have been described as without pathological changes. Nevertheless, upon more careful examination by means of Lenhossek's and Nissl's methods, distinct degenerative changes were found in the motor cells, as shown by the disappearance of the chromophilic granules of the protoplasm and the changes in the nucleus and nucleolus. In both cases degenerative changes were found in the axis-cylinders of the peripheral nerves. Probably in these cases we have again to do with a degenerative process confined to the motor neuron of the first order produced by an intoxication of some sort. We must admit that similar changes have been found both in the cells and in their processes, in cases which clearly belong to polyneuritis. The fact that the course of Landry's paralysis differs so markedly from an ordinary case of neuritis, that the sensory neuron is so little if at all affected, and that the tenderness of the nerve-trunks to pressure is absent or slight, seems to justify us in retaining this collection of symptoms as forming a separate disease, clinically at least.

If this conception of Landry's paralysis as a degenerative process of the peripheral motor neuron, with or without the presence of an exudative inflammatory process in the anterior horns of the cord, be correct, the absence of the reaction of degeneration in the muscles affected can only be explained by the short duration of the disease, and the same is true of the absence of atrophy; so that one is compelled to attach less importance to the absence of these symptoms than has hitherto been done, while the absence of sensory changes acquires increased importance.

Analysis of the reported cases as to the cause shows that in a great many none could be ascribed, nevertheless in a considerable number of cases acute ascending paralysis followed closely upon some infectious disease, such as smallpox, diphtheria, or typhoid. The presence of an enlarged spleen and of swelling of the mesenteric lymph glands, which is so often noted in the autopsies, confirms this view. It has been said that we may expect to find some micro-organisms present in the nervous structures, but more often it is probable that the micro-organisms producing the poison will be found in some other part of the body. Inasmuch as nerve-cells react in much the same way to various poisons, further research will probably show that in these cases micro-organisms are not always present, but that the intoxication may be produced through faulty metabolism or by the absorption of poisons from without. That toxic substances may act in a selective manner, affecting only the motor neurons, is difficult to explain, although we are not without other instances of the same action, as, for example, the almost pure motor trouble in lead paralysis, which, in this case, as is well known, has a special predilection for the motor nerves and for those going to the extensor muscles of the forearm.

THE USE OF ANIMAL TOXINS IN THE TREATMENT OF INOPERABLE MALIGNANT TUMORS.[1]

By George Ryerson Fowler, M.D.,
PROFESSOR OF SURGERY, NEW YORK POLYCLINIC; SURGEON-IN-CHIEF, BROOKLYN HOSPITAL; SURGEON TO THE METHODIST EPISCOPAL HOSPITAL.

THE CURE OF MALIGNANT DISEASE BY ACCIDENTAL ERYSIPELAS. Early in the seventeenth century, according to Fehleisen, it was known that not only malignant growths, but certain chronic ulcers, lupus and syphilitic destructive lesions, were arrested, and in some instances absolutely disappeared following an invasion of erysipelas. In modern times Billroth called attention anew to the subject, by reporting a case of inoperable sarcoma of the pharynx cured by an attack of facial erysipelas.

There has been some dispute as to the authenticity of such cases, but it must be admitted that while it is very difficult to verify at this late day the assertion that all the cases denominated true sarcoma were in reality examples of that disease, yet it is going too far to say that all of the accounts bearing upon the question are absolutely untrustworthy. Nor will it suffice to declare the occurrence of the erysipelas and the disappearance of the growths as merely coincidental. The theory of spontaneous disappearance of the growths as a coincidence in the course of or following an attack of erysipelas falls at once to the ground in the face of the fact that the spontaneous disappearance of growths which, even with the limited knowledge of pathology at the command of our predecessors, could have been mistaken for malignant disease, is absolutely unknown. Moullin,[2] in investigating this subject in a most conservative, painstaking, and impartial manner, was able to identify fifteen undoubted instances of sarcoma in which an attack of erysipelas occurred. Of these, in no less than nine all evidences of the growth disappeared, and in some instances no recurrence had taken place for seven years. As corroborative evidence of the influence of the erysipelas it may be said that, of the remaining six cases, five showed a decided lessening in the size of the growth.

The cases reported by Billroth,[3] Busch,[4] Biedert,[5] Plenno,[6] Bruns,[7]

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[1] Presented at the meeting of the American Surgical Association at New Orleans, April 20, 1898.

[2] The Lancet, February 5, 1898.

[3] Senn: The Journal of the American Medical Association, July, 1895, p. 131.

[4] Berliner klinische Wochenschrift, 1866, No. 23.

[5] Deutsche med. Zeitung, 1895, No. 4.

[6] Archiv f. klin. Chirurg., 1886, Bd. xxxiv. p. 688.

[7] Beitrage zur klin. Chirurg.